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Association of physical and sexual assault with mortality in two British birth cohorts

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ABSTRACT

Aims The association of assault in adulthood with all-cause mortality, and the relevance of intermediate psychological distress, alcohol use and cigarette smoking, is poorly understood. We used data from British birth cohorts (the 1958 National Child Development Study referred to as the 1958 birth cohort and the 1970 British Birth Cohort Study) to investigate association between assault and mortality, employing a formal approach for the identification of psychological distress, alcohol use and cigarette smoking as mediators.

Methods Associations (HRs), between assault and mortality were estimated with Cox regressions, adjusting for potential confounders. Mediation via intermediate psychological distress, alcohol use and cigarette smoking was explored using the gformula approach. The birth cohorts were analysed separately, and together estimating interaction between exposure and cohort year.

Results Results were based on 353 deaths in 19725 individuals. Based on multiply imputed data, the fully adjusted estimate for assault on mortality was 1.72 (95% CI 1.22 to 2.42) in the combined cohorts, 1.53 (95% CI 0.97 to 2.40) in the 1958 birth cohort and 2.05 (95% CI 1.20 to 1.50) in the 1970 birth cohort. The fully adjusted estimate for the association of sexual assault with mortality was 3.17 (95% CI 1.17 to 8.60) in the combined cohorts, 1.36 (95% CI 0.19 to 9.81) in the 1958 birth cohort and 6.02 (95% CI 1.84 to 19.69) in the 1970 birth cohort. The fully adjusted mortality HR for one additional assault was 1.46 (95% CI 1.23 to 1.73) in the combined cohorts, 1.34 (95% CI 0.99 to 1.82) in the 1958 birth cohort and 1.53 (95% CI 1.25 to 1.87) in the 1970 birth cohort. Greater need for medical treatment for assault was associated with a fully adjusted mortality HR of 1.56 (95% CI 1.19 to 2.05) in the combined cohorts, 1.43 (95% CI 1.00 to 2.05) in the 1958 birth cohort and 1.79 (95% CI 1.18 to 2.74) in the 1970 birth cohort.

Conclusions There was statistical evidence on combining the two birth cohorts, and on analysing the 1970 birth cohort, that assault in adulthood is associated with mortality. Understanding mechanisms underlying this relationship could benefit violence reduction strategies for public health.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Physical and sexual assault are known to affect health, but the association with all-cause mortality has seen limited research.

WHAT THIS STUDY ADDS

⇒ This study quantifies the impact of assault exposure for population mortality. We find strong evidence for association between assault exposure and mortality in two British birth cohorts, a dose-relation with number of assaults, and a greater mortality impact of more severe assaults.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Improving research evidence on the mechanisms underlying the relationship between assault exposure and mortality could benefit violence prevention strategies.

INTRODUCTION

Interpersonal violence, including physical and sexual assault, is a complex global challenge.[1] Around 1 in 50 British adults experienced violent crime in 2018, and the cost of violent crime in Britain is estimated at £20 billion per year.[2 3] Consistent inequalities are evident in the population distribution of violence, with a greater incidence of violence among young men,[4 5] and those exposed to adverse childhood experiences, socioeconomic disadvantage and alcohol use.[6] The incidence of assault is higher among people with lower cognitive performance (compared with those with higher performance),[7] and people with pre-existing depressive symptoms and psychological distress.[8–10]

Effective violence reduction strategies are a policy priority in high-income countries, however, the potential impact, and underlying mechanisms, of violence reduction strategies on public health remains unclear. There have been limited attempts to estimate the impact of violence on population health, or to assess pathways by which violence reduction strategies might achieve population health benefit. Quantifying this effect could be relevant for

improving the impact of population violence prevention strategies on health, and for identifying new mechanisms for population health improvement.

Previous work has estimated the association of assault with general mortality but did not assess mediating explanations.[11] For example, psychological distress and morbidity are also a common consequence of physical and sexual assault,[12] and may in turn also be linked to higher mortality risk[13–16] although there is some inconsistency in these findings.[17 18] As well as being associated with assault exposure, alcohol intake is increased as a consequence of physical[19 20] and sexual assault[21]; and a consistent association of alcohol use with general mortality has been established for more than two decades.[22 23] Cigarette smoking has been associated with prior assault in epidemiological studies[24 25] and remains a strong driver of premature mortality.[26 27]

While there is evidence of some fluctuation in the general population incidence of assault over time,[5] a relationship between assault exposure and morbidity remains persistent; differences in any association between assault and mortality over time has not been tested and could be relevant for explaining time trends in mortality in the general population. This could be helpful in developing refined interventions for people exposed to assault in healthcare settings. Gender differences in the incidence of assault are evident, especially when considering hospitalisation data,[4 5] but there is mixed evidence on gender differences in the impact of interpersonal violence including assault on longer-term health outcomes, including mortality.[28–30] We have found evidence for a relationship between violence in childhood and increased risk of experiencing violence in adulthood,[31] in line with wider evidence for clustering of violent experiences within individuals, and emphasising the need to understand the impact of cumulative violence exposure on health and well-being.[32 33]

The impact of adulthood exposure to physical/sexual assault on mortality, and the role of psychological distress, or intermediate alcohol use, or intermediate cigarette smoking, in explaining or shaping such a relationship, is poorly characterised. The goal of this study was to investigate the association between assault and mortality. Our objectives were to:

a. Estimate the association between assault and all-cause mortality.
b. Evaluate psychological distress, alcohol use and cigarette smoking as mediators of this association.

We carried out our study using data from birth cohorts started in 1958 and 1970, to further examine moderation of the relationship between assault and all-cause mortality by birth cohort.

METHODS

Cohort details

Data for this study were from the 1958 National Child Development study (NCDS, referred to as the 1958 birth cohort hereafter), a birth cohort collecting data on all live births taking place in a single week in 1958 in England, Wales and Scotland[34]; and the 1970 British Cohort Study (BCS, the 1970 birth cohort hereafter), a birth cohort collecting data on all live births taking place in England, Scotland, Wales and Northern Ireland in a single week in 1970.[35] Measurements on all variables in this study, including ages of measurement, are summarised in figure 1. A flow diagram showing the study sample was arrived at is displayed in figure 2.

Measurements

Mortality

Data on mortality were taken from the NCDS Deaths Dataset, containing data on known deaths in the 1958 birth cohort occurring from 1958 to 2014, and the BCS Deaths Dataset, with data on known deaths in the 1970 birth cohort occurring 1970–2014. Each dataset was compiled using records maintained by organisations responsible for the two studies over their lifetimes: the National Birthday Trust Fund, the National Children's Bureau, the Social Statistics Research Unit and the Centre for Longitudinal Studies. Sources for mortality data in both cohorts included death certificates and other information from the National Health Service Central Register, and from relatives and friends during survey activities and cohort maintenance work by telephone, letter and e-mail. The analysis of mortality used only deaths occurring from 2000 to 2014, because of the measurement point for assault. Data were shared by the UK Data Service in 2017, and main analyses were undertaken from 2018 to 2020.

Assault

We made use of identically worded questions measuring assault exposure in the two cohorts. In the 1958 birth cohort, assault data were collected in two consecutive waves: self-report information on assaults occurring since age 25 was collected at age 33, in 1991, and further

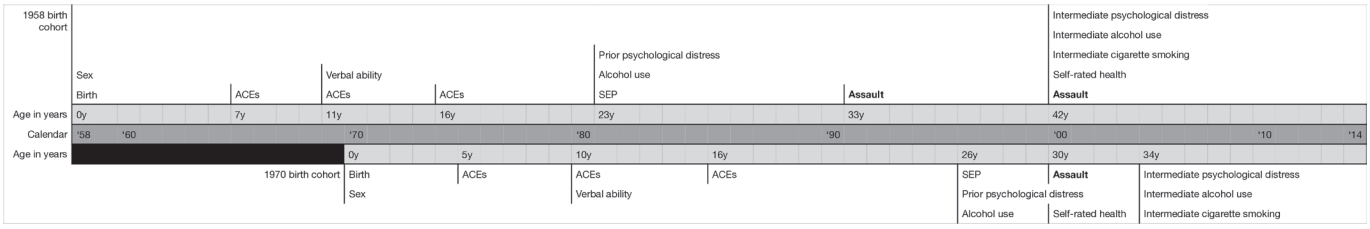

Figure 1

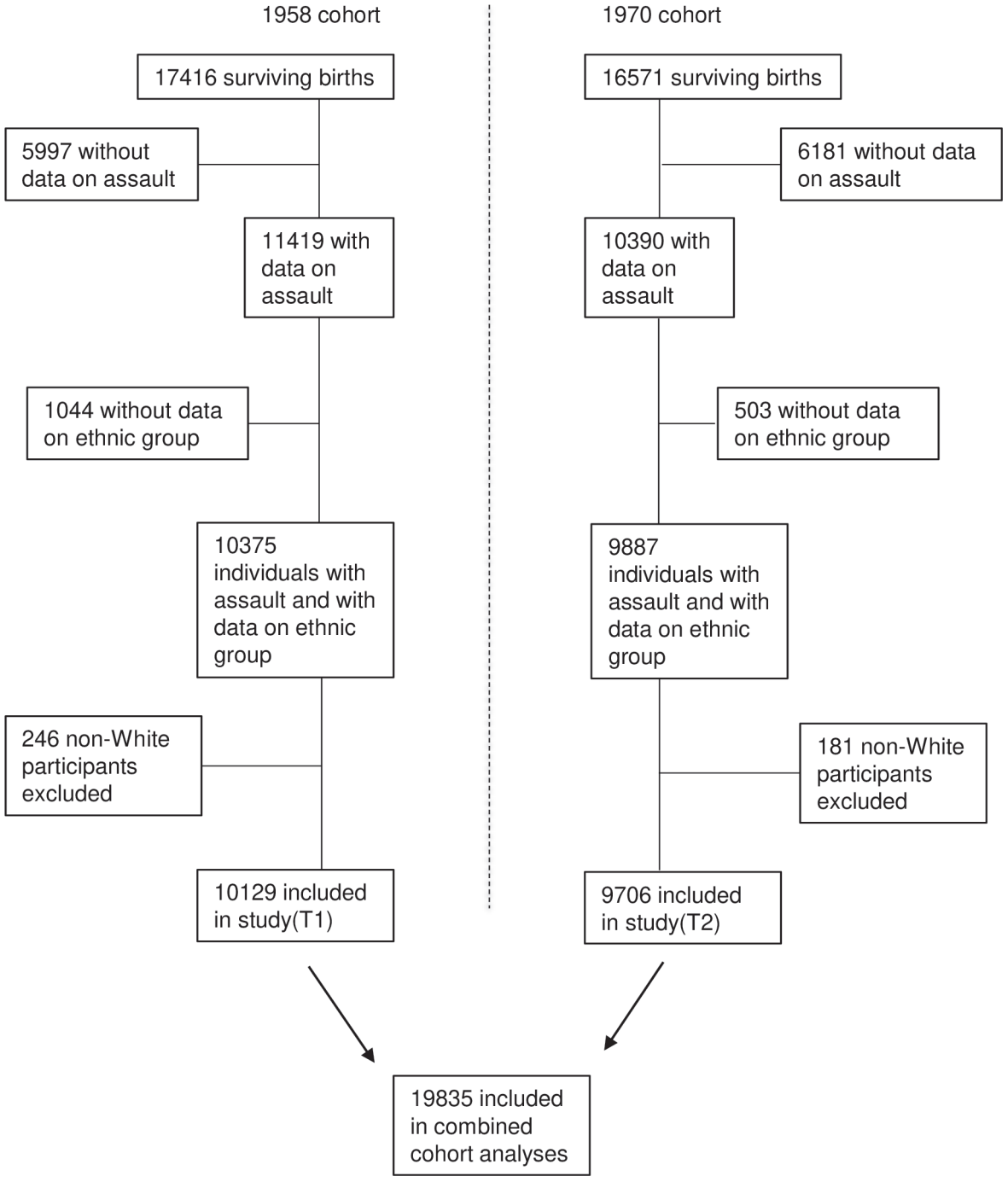

Figure 2

self-report information on assault occurring since age 33 was then collected at age 42, in 2000. In the 1970 birth cohort, self-report information on assaults occurring since age 21 was collected at age 30, in 2000. As described below, sensitivity analyses assessed the impact of the longer overall reference period in 1958 birth cohort members, on our results.

In both birth cohorts, information on assault was gathered by asking participants if they had since the reference date, received medical attention for a physical or sexual assault. Relevant responses to this item were used to generate a binary variable for any (physical or sexual) assault within the reference period, and for physical and sexual assaults separately. Data on the total number of physical or sexual assaults experienced in the reference period were also collected. Self-report information was used to generate a three-level variable for highest level of medical treatment received for assault, classified into no assault, medical treatment but not overnight and overnight medical treatment.

Measurement of mediators

To assess psychological distress as a potential mediator of the association between assault and mortality, we

included similar data on psychological distress based on measurements collected on the Malaise Inventory at age 42 in 2000 (1958 birth cohort) and 34 in 2004 (1970 birth cohort). To measure alcohol, units of alcohol consumed per week were taken at age 42 in the 1958 birth cohort and age 34 in the 1970 birth cohort, using conventional formulae for converting self-reported weekly consumed quantities of different beverages (beer, wine, shandy, sherry, spirits and alcopops (1970 birth cohort only)) to units of alcohol consumption.[36] To measure cigarette smoking, we used measurements of current numbers of cigarettes smoked at age 42 in the 1958 birth cohort and age 34 in the 1970 birth cohort. Measurement of covariates is described in the methodological supplement.

Analysis

Data were analysed by using Stata V.17.[37] Individuals with missing data on assault were excluded. Counts and proportions of cohort members experiencing any assault, and the number of total assaults, were described by covariates for each birth cohort separately. To account for confounding, adjustment variables were selected for inclusion based on a review of the literature for each characteristic's influence on both assault and mortality. A directed acyclic graph displaying pathways underlying the analysis is shown in online supplemental figure 1. Data on continuous variables (alcohol use, cognitive (verbal) ability) from both birth cohorts were z-standardised to have a mean of 0 and an SD of 1, and all were included as linear terms after assessment of difference in goodness of fit versus quadratic terms and indicator terms for each quintile (measured by the Akaike information criteria and the Bayes information criteria, see online supplemental table ST1). We used information on how many years ago each assault occurred to derive a variable for age of assault, describing this in both cohorts (online supplemental table ST2).

To test any association between assault and mortality, Cox regression models were estimated including 1 January 2000 as the start of the at-risk period, defining death as the time-to-event outcome and censoring defined by being unavailable for follow-up at any wave of data collection, or the conclusion of the time-at-risk. Graphical inspection of log/log plots (see online supplemental figure 2) and of Schoenfeld residuals were used to check departure from the assumption of proportionality of hazards. Kaplan-Meier graphs for each cohort stratified by any assault are displayed in online supplemental figure 3 (1958 cohort) and 4 (1970 cohort). Models were estimated for any assault (ie, either physical or sexual assault), physical assault, sexual assault, a count variable for the number of assaults, and highest level of medical treatment for any assault (classified into no assault, medical treatment but not overnight and overnight treatment). All analyses were carried out on data combining the two birth cohorts, and on each birth cohort separately—we also tested for interaction by cohort year. To report the impact of adjustments for different confounders, we estimated the crude association (model I), models only adjusting for gender, adverse childhood experiences and birth year only (model II), then further adjusting for socioeconomic variables (marital status, class, educational attainment, model III) and then further adjusting for model III variables and prior psychological distress, verbal ability and alcohol use (model IV, the final model). For adjusted analyses combining both cohorts, we adjusted for adverse childhood experiences using the restricted definition of adverse childhood experiences (excluding neglect) to ensure uniformity of definitions between the cohorts. For analyses of 1958 birth cohort data, we estimated models with both definitions of adverse childhood experiences (including and excluding neglect). To assess the impact of differing reference periods for assault measurement on our results, combined cohorts analyses were estimated excluding either the early or the late reference period for 1958 birth cohort data (presented in online supplemental table ST3). Based on the final model for the combined cohorts, we tested for differences between men and women, by the presence of prior psychological distress, and by cohort year, using multiplicative interaction terms, setting $p \leq 0.05$ as the criterion for determining significant interactions.

To address the impact of missing data, we also estimated the same models based on 10 multiply imputed datasets, using the mi command in Stata, which deploys multiple imputation with chained equations. Imputation commands for each missing variable were specified based on their form in complete case models—imputation models contained: sex, marital status and prior psychological distress, imputed by logistic regression; adverse childhood experiences, social class and educational attainment, imputed by ordinal logistic regression and cognitive (verbal) ability and alcohol use imputed by linear regression. Directions of associations remained after accounting for missing data in imputations (based on the missing at-random assumption).

In addition to Cox models, we used the gformula package to explore mediation.[38] According to a counterfactual framework, G formula analyses contrast-specific exposure (or treatment) scenarios—in this case, comparing a scenario where the entire population is assigned to no exposure (eg, no assault), with the scenario where the population is assigned to exposure (assault). To examine mediation of the association between assault and mortality by the candidate mediators (psychological distress, alcohol use and cigarette smoking), we estimated total controlled effects, direct effects (DE) and natural indirect effects (NIE) for any assault. The DE represented the effect of assault on mortality that was independent of each respective mediator. The NIE represented the proportion of mortality that could be explained by its association with changes in each respective mediator over time. To quantify the magnitude of mediation, we estimated the proportion of the association mediated (NIE/[DE+NIE]). G formula models further adjusted for

general health status as a postbaseline mediator-outcome confounder (as shown in online supplemental figure 1). For missing data, the gformula package implements single stochastic imputation using chained equations. We included identical specifications of each variable in G formula models as in Cox models described above.

Patient and public involvement

Patients or the public were not involved in the design, or conduct, or reporting, or dissemination plans of our research.

RESULTS

Description

The 1958 birth cohort

Online supplemental ST4 describes 1958 birth cohort members of white ethnicity with available data on assault (n=10 129). Mortality among females was 2.0%, and among males, it was 2.9%. Greater mortality was associated with adverse childhood experiences, being in the lowest occupational class, having no qualifications and single, divorced or widowed marital status. Mortality was higher among individuals reporting poor health and among those with psychological distress. Those who died also had lower standardised scores for cognitive (verbal) ability and higher standardised scores for alcohol use.

In the 1958 birth cohort, experiencing assault was more common among men compared with women, and among those experiencing each adverse childhood experience except for parental alcohol use, where the proportion who reported assault (4.6%) was similar to that among those reporting no parental alcohol use (4.5%). A greater prevalence of assault was associated with being in the lowest occupational class, having no qualifications, reporting poor general health, lower verbal ability, higher alcohol use and with reporting psychological distress.

The 1970 birth cohort

Online supplemental ST5 describes 1970 birth cohort members of white ethnicity with available data on assault (n=9706). Mortality among males was 1.5% compared with 0.8% in females. Mortality was generally similar among those experiencing each adverse childhood experience compared with others. Mortality was similar by educational status and by verbal ability. Standardised scores for alcohol use were higher among those who died.

In the 1970 birth cohort, experiencing assault was commoner among men compared with women, among those experiencing each adverse childhood experience, among the lowest occupational class and among those with no educational qualifications. Prevalence of assault was also higher among those with fair/poor general health and among those with psychological distress. Standardised scores for alcohol use were higher among those who experienced assault.

Association of assault and mortality

Figure 3 graphically displays multiple imputation-based estimates for the association of assault with mortality; a table of partially adjusted estimates is reported in online supplemental table ST6. Based on multiply imputed data, the fully adjusted estimate for assault on mortality was 1.72 (95% CI 1.22 to 2.42) in the combined cohorts, 1.53 (95% CI 0.97 to 2.40) in the 1958 birth cohort and 2.05 (95% CI 1.20 to 1.50) in the 1970 birth cohort (see figure 3). Results for physical assault specifically were similar to any assault in direction and magnitude. The fully adjusted estimate for the association of sexual assault with mortality was 3.17 (95% CI 1.17 to 8.60) in the combined cohorts, 1.36 (95% CI 0.19 to 9.81) in the 1958 birth cohort and 6.02 (95% CI 1.84 to 19.69) in the 1970 birth cohort. The fully adjusted mortality HR for one additional assault was 1.46 (95% CI 1.23 to 1.73) in the combined cohorts, 1.34 (95% CI 0.99 to 1.82) in the 1958 birth cohort and 1.53 (95% CI 1.25 to 1.87) in the 1970 birth cohort. Greater need for medical treatment for assault was associated with a fully adjusted mortality HR of 1.56 (95% CI 1.19 to 2.05) in the combined cohorts, 1.43 (95% CI 1.00 to 2.05) in the 1958 birth cohort and 1.79 (95% CI 1.18 to 2.74) in the 1970 birth cohort. Estimates from complete-case analysis are reported in online supplemental table ST7.

Interactions

No statistical evidence was found for interaction by gender (interaction HR 1.14 (95% CI 0.21 to 6.11), p=0.877), by cohort year (interaction HR 0.52 (95% CI 0.09 to 2.80), p=0.442) or by prior psychological distress (interaction HR 0.68 (95% CI 0.15 to 3.14), p=0.625). Mediation results are reported in online supplemental table ST8 and described in the methodological supplement.

DISCUSSION

Summary of findings

We report the first prospective examination of association of exposure to physical or sexual assault in adulthood, with all-cause mortality in data from two British birth cohorts. We observed strong mortality associations for exposure to any assault, physical assault specifically, an increasing number of assaults and an increasing severity of assault (as indicated by the level of medical treatment received), on analysis of the combined cohorts. Statistical evidence for association of sexual assault, specifically, with mortality was incomplete due to small numbers of individuals reporting sexual assault. We did not find evidence for mediation of this association by intermediate psychological distress, alcohol use or cigarette smoking.

Limitations and strengths

We made use of detailed self-report information on exposure to physical and sexual assault. Final estimates for our main comparison, the association of assault with mortality, were statistically significant and based on large nationally representative British birth cohorts. We demonstrated trends in associations with assault number, and with assault severity (as indexed by degree

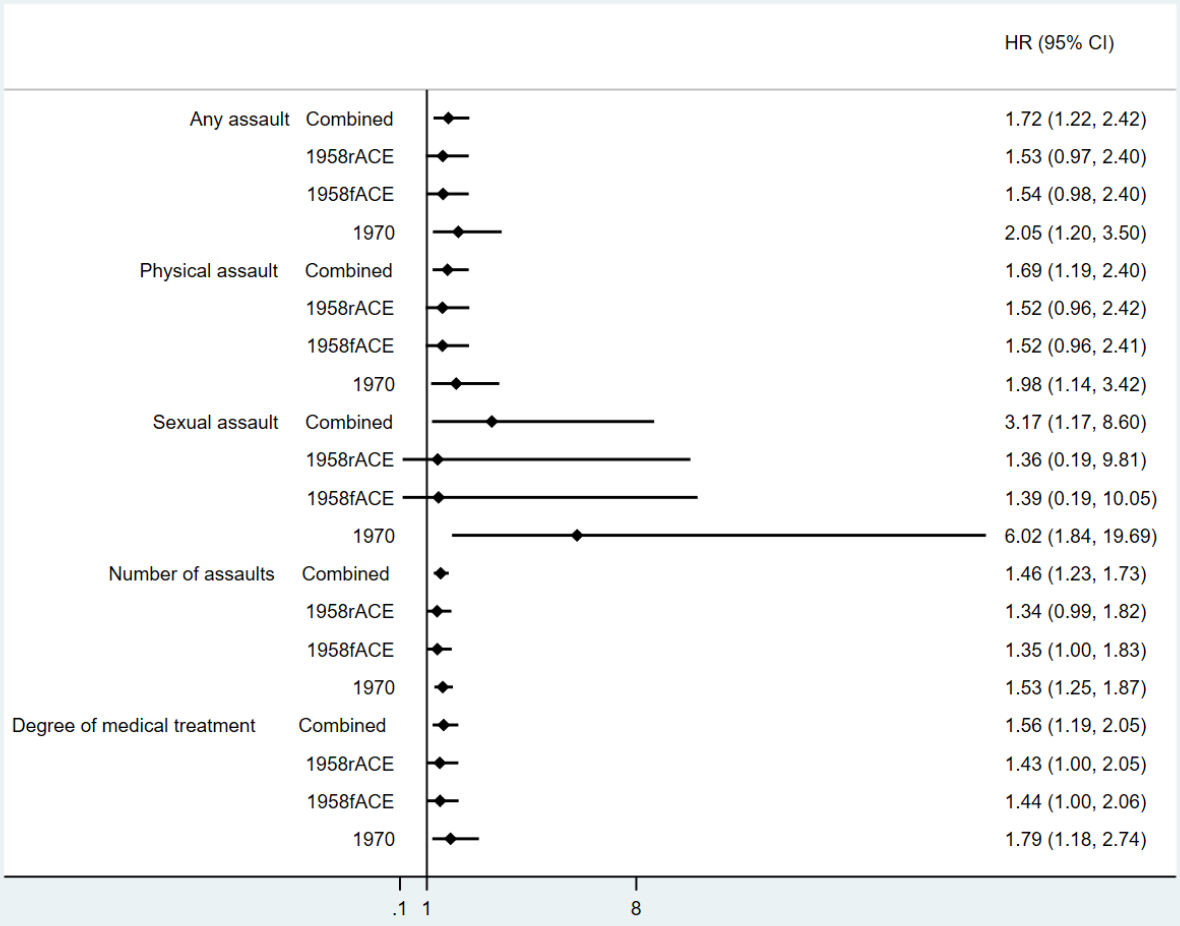

Figure 3 Diagram displaying fully adjusted model estimates (HRs) from multiple imputation for the association of any assault, physical assault, sexual assault, number of assaults and need for treatment for assault, with mortality. Estimates are adjusted for sex, cohort year, ACEs, socioeconomic variables (class, educational attainment and marital status), and alcohol and cognitive/psychiatric covariates (alcohol use, cognitive (verbal) ability and prior depressive symptoms). (A) Based on data from 19 725 individuals. (B)1958rACE refers to estimates for the 1958 birth cohort employing a restricted definition of ACEs (leaving out neglect) to allow comparison with 1970 birth cohort estimates. Based on data from 10 019 individuals. (C)1958fACE refers to estimates for the 1958 birth cohort employing the full definition of ACEs (including neglect). Based on data from 10 019 individuals. (D) 1970 birth cohort estimates were based on data from 9706 individuals.

of medical treatment), strengthening inferences of association between assault exposure and mortality. To our knowledge, we present the first epidemiological description of assault data in these birth cohort studies—with age distributions for assault exposure being consistent with existing evidence suggesting greater occurrence of assault exposure in younger age groups.[6]

Data on the location of assaults, and relationship of individuals exposed to assault to the perpetrator, were not collected. Informative censoring, for example, through greater censoring of individuals at higher risk of the both the exposure and the outcome could have resulted in bias in our estimates, probably in the null direction. Data on individuals who experienced assault and then died before the start of observation time were not used, which may have introduced bias towards the null through omission of individuals with shorter failure times. Use of self-report data on assault could have introduced reporting bias, if, for example, the probability of reporting assault was related to mortality risk, however, main drivers of mortality are unlikely to have affected recall. Other methods of measuring assault, such as direct use of linked hospital records, could have generated more temporally precise data. Our study examined individuals who sought medical attention for assault rather than the larger population exposed to any assault. Although we adjusted for theoretically guided confounders, residual confounding by unknown common causes of assault and mortality remains possible, including for indirect effects. We excluded non-white individuals from our analysis because of very small numbers of exposed individuals from minority ethnic groups, limiting the generalisability of our results. The use of two birth cohorts, collected 12 years apart, allowed testing for cohort differences in associations, but also involved the pooling of assaults data collected during different life periods (age 25–42 in 1958

birth cohort and age 21–30 in the 1970 birth cohort), limiting our ability to assess the impact of age of exposure on our results. Although crude estimates for the association of sexual assault with mortality suggested a statistically significant association, fully adjusted estimates were statistically imprecise, indicating that confounding cannot be excluded as an explanation for this association based on current evidence. We estimated a number of statistical comparisons, which likely increased the likelihood of type I error. However, these were prespecified and our estimates of association were consistent in terms of direction across the different comparisons.

Finally, differences in estimates from multiply imputed data from estimates drawn from complete-case analysis suggests bias introduced by missing data when considering analyses of complete cases.

Explanations

Our previous work indicated association of physical assault with mortality in men living in Russia[11] but was unable to examine intermediate explanations. Kelly-Irving *et al*[39] report the influence of adverse childhood experiences on mortality but did not examine the role of exposure to assault in adulthood. Participants in a longitudinal study of Danish adults who reported the highest frequency of relationship conflict (including with partners, family members, coworkers and neighbours) experienced significantly greater mortality. This analysis did not examine violence specifically, limiting its implications for violence specifically.[40] Our findings make a further contribution to evidence on violence exposure and population health.

The evidence we found for a relationship between count of assaults and mortality implies a role for accumulated experience of interpersonal violence in adulthood and mortality through the 'wear and tear' of body systems, as proposed in the allostatic load hypothesis.[41] Under the allostatic load hypothesis, the accumulation of stressful experiences, including violence, could result in chronic activation of physiological stress responses, including the hypothalamo–pituitary–adrenal axis, driving inflammatory and degenerative processes and increasing mortality, for example, through cardiovascular disease.[41]

CONCLUSIONS

Effective violence reduction strategies can improve population health, as indexed by all-cause mortality. This effect might work through an improvement in psychological distress in the population, reduced alcohol use or reduced cigarette smoking, however, this requires evaluation in larger datasets. Substantial excess all-cause mortality in people exposed to physical or sexual assault provides direct evidence supporting violence prevention policies for population health improvement.

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Contributors VB conceived the study and analysed the data. WL advised on study design. All authors reviewed, edited and commented on draft manuscripts.

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Disclaimer The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1965, as revised in 2008.

Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants and the 1958 birth cohort obtained ethical approval from the South East MREC (ref:01/1/44). Ethical approval for the 1970 birth cohort was obtained from a National Health Service Research Ethics Committee in advance of each sweep of data collection. For example, the 2008 survey was approved by Southampton and South West Hampshire Research Ethics Committee (08/H0504/144). Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data may be obtained from a third party and are not publicly available. Data used in this study are available for use for research on application to the UK Data Service (https://ukdataservice.ac.uk/).

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